CASE REPORT

Cardiopulmonary arrest owing to oesophageal achalasia recovered completely with cardiopulmonary resuscitation followed by therapeutic hypothermia

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SUMMARY

We report the first case of cardiopulmonary arrest (CPA), caused by oesophageal achalasia, which recovered completely with cardiopulmonary resuscitation (CPR) followed by therapeutic hypothermia. A 53-year-old woman arrived at our hospital with recovery of spontaneous circulation (ROSC) after cardiac arrest. Dysphagia, vomiting and general fatigue had progressed for a week before. After an ambulance was called for severe dyspnoea, she collapsed in CPA. Emergency medical technicians arrived and CPR was started immediately. She experienced CPA and ROSC twice during transport to the hospital. On arrival, the patient was in respiratory distress prompting immediate intubation to eliminate airway obstruction. A CT scan revealed a transformed, occluded trachea owing to a dilated oesophagus. A large amount of food, air and saliva was suctioned with a nasogastric tube, and the patient was admitted to the intensive care unit for therapeutic hypothermia. Neurological recovery was evident. On the 11th day, pneumatic dilatation was performed and she was discharged on the 33rd day.

BACKGROUND

Achalasia is a neurodegenerative condition leading to failure of peristalsis, with raised pressure and incomplete relaxation in the lower oesophageal sphincter during swallowing. Respiratory obstruction owing to tracheal compression caused by a massively dilated oesophagus is a very rare but fatal condition.¹²

This is the first case of cardiopulmonary arrest (CPA) owing to airway obstruction caused by oesophageal achalasia, which was completely recovered with cardiopulmonary resuscitation (CPR), followed by therapeutic hypothermia.

CASE PRESENTATION

A 53-year-old woman was transported to our hospital with recovery of spontaneous circulation (ROSC) after CPA because of airway obstruction. After calling an ambulance for severe dyspnoea, she collapsed in CPA. The initial cardiac rhythm was pulseless electrical activity (PEA) confirmed by emergency medical technicians (EMTs), and CPR, including chest compression and bag-valve-mask ventilation, was started immediately. She experienced CPA (PEA) and ROSC twice during transport to the hospital, and ROSC was obtained after

2 min of CPR in both instances. Epinephrine administration during CPR was not performed by EMTs because EMTs are not allowed to give injections in Japan.

Her medical history was significant for asthma and her family medical history was unremarkable. She had developed dysphagia 13 years previously but was not thoroughly examined at the time. Dysphagia, vomiting and general fatigue had progressed for 1 week prior to admission to our hospital.

On arrival, the patient was in respiratory distress with a Glasgow Coma Scale (GCS) score of 3/15. Her vital signs were as follows: body temperature, 37.4°C; blood pressure, 110/71 mm Hg; heart rate, 109 beats/min and respiratory rate, 30 breaths/min. Her oxygen saturation was 99% while breathing 10 l/min of oxygen via a non-rebreather mask. Bilateral jugular veins were markedly distended and a subcutaneous swelling in the left side of the neck was noted. Auscultation revealed stridor during inspiration. Her pupils were 5 mm in diameter, equal in size and round. The rest of the examination was unremarkable.

Complete blood cell counts initially showed a white blood cell count of 14 500/μl; haematocrit, 43.5% and platelet count, 179 000/μl. Arterial blood gas analysis performed while the patient was breathing 10 l/min of oxygen via a non-rebreather mask showed pH, 7.923; PCO₂, 134.2 mm Hg; PO₂, 167.8 mm Hg; HCO₃⁻, 27.1 mmol/l and oxygen saturation, 98%.

INVESTIGATIONS

ECG showed normal sinus rhythm and non-specific ST-T wave changes.

A chest x-ray revealed an oesophageal gas shadow in the left side of the neck, an enlarged upper mediastinal outline and an ill-defined trachea border (figure 1). A CT scan showed a transformed and occluded trachea owing to a dilated oesophagus, with no acute abnormalities in the head (figures 2A–D and 3A,B). An echocardiogram exposed diffuse left ventricular wall hypokinesis.

Upper gastrointestinal endoscopy demonstrated a massively dilated lumen of the cervical oesophagus and a large amount of undigested food. The endoscope was able to pass through the oesophagogastric junction with no apparent stenosis of the lower

To cite: Hifumi T, Okada I, Inoue J, et al. *BMJ Case Reports* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008378

Figure 1 A chest x-ray on admission showed an oesophageal gas shadow in the middle of the neck, enlarged upper mediastinal outline, and ill-defined tracheal border.

oesophageal sphincter. There was no evidence of oesophageal or stomach malignancies.

DIFFERENTIAL DIAGNOSIS

Airway obstruction owing to oesophageal achalasia was suspected to have caused CPA. Cardiomyopathy was also considered as a possible cause owing to poor left ventricular wall motion.

TREATMENT

Intubation for airway obstruction was performed immediately on admission. The supraglottic airway and motility of the vocal cords were normal. Intravenous access was obtained. A large amount of food, air and saliva were suctioned using a nasogastric tube. Haemodynamic optimisation was attained with intravenous dopamine and dobutamine. The patient was cooled initially with 1.5 litres of normal saline and was admitted to the intensive care unit for further treatment including therapeutic hypothermia. She was cooled to a target temperature of 32–34°C using a

Figure 3 A chest CT scan showing a coronal view of (A): subcutaneous gas in the left side of the neck is shown by the arrow and (B) abnormal dilatation of the oesophagus caused by food and air is shown by the arrow.

mattress with a cover that delivered cold air over the entire body. The goal was to reach the target bladder temperature within 4 h after ROSC. Her temperature was maintained at 32–34°C for 24 h from the start of cooling, followed by passive rewarming, and expected to occur over a period of 8 h. Once normothermia was attained, sedation was discontinued.

OUTCOME AND FOLLOW-UP

Neurological recovery was evident and extubation was performed after decompression of the oesophagus by upper gastrointestinal endoscopy. She was moved to the general ward on the 10th day of admission, and pneumatic dilatation was performed for achalasia on the 11th day. The remainder of her hospital stay was uneventful, and she was discharged on the 33rd day without any neurological complications.

DISCUSSION

Bello *et al*³ first reported acute upper airway obstruction caused by achalasia in 1950. To date, approximately 40 such cases have been described in the literature.⁴ Moreover, three reports have

Figure 2 A chest CT scan showing an axial view of (A–C) arrows indicate a transformed and occluded trachea owing to a dilated oesophagus and (D) the narrowest portion of the oesophagus is marked by an arrow.

detailed four instances of sudden death caused by achalasia.^{5–7} However, a case of CPA owing to acute upper airway obstruction caused by oesophageal achalasia that recovered completely has not been published. In the present case, the patient recovered fully after CPR followed by therapeutic hypothermia.

Clinical features of cases with airway obstruction caused by oesophageal achalasia are similar, in that most of the patients were elderly women and symptoms generally start postprandially.²⁸ The reason why elderly women develop airway obstruction because of achalasia remains unclear. Tracheobronchomalacia owing to a longer period of compression by a dilated oesophagus is considered as a possible cause⁸; however, in the present case, the patient was middle-aged and tracheobronchomalacia was not observed.

The patient called an ambulance late at night at 23:00 h but had not complained of respiratory distress soon after eating. As a result of these two details, the present case was considered an unusual clinical presentation, hindering a prompt diagnosis and emergency treatment.

Therapeutic hypothermia is now the standard of care for adult patients with ROSC who remain comatose following an out-of-hospital ventricular fibrillation cardiac arrest. This treatment might also be beneficial after non-shockable or in-hospital cardiac arrest^{9–10}; however, the literature related to the application of therapeutic hypothermia, specifically for comatose survivors of airway obstruction, including near-hanging,¹¹ submersion¹² and advanced laryngeal cancer, is limited.¹³ Lee *et al*¹⁴ concluded that outcomes in unconscious near-hanging patients with CPA were poor despite treatment with therapeutic hypothermia because of the more severe nature of ischaemic brain injury after cardiac arrest owing to asphyxia than from ventricular fibrillation.

We provided therapeutic hypothermia for this patient because the patient's GCS score was 3/15 on admission. However, the efficacy of therapeutic hypothermia with this patient was not confirmed because this patient developed CPA for a short time.

In conclusion, oesophageal achalasia could cause airway obstruction. The fact that this condition is not limited to elderly women should be considered. Further large studies are required to evaluate the efficacy of therapeutic hypothermia for patients with CPA owing to airway obstruction.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- Oesophageal achalasia could cause airway obstruction.
- Airway obstruction caused by oesophageal achalasia is not limited to elderly women.
- Further large studies are required to evaluate the efficacy of therapeutic hypothermia for patients with cardiopulmonary arrest owing to airway obstruction.

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